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Clinical Images

Central pontine myelinolysis

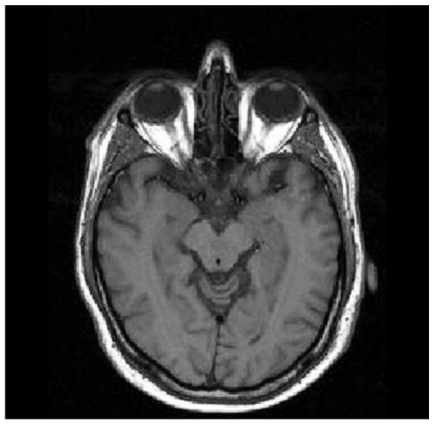

Fig. 1. T1 weighted MR image of the brain.

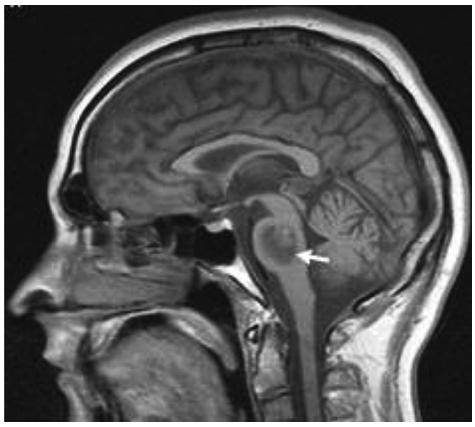

Fig. 2. Brain MRI of the patient showing a low T1 signal intensity lesion (day 14).

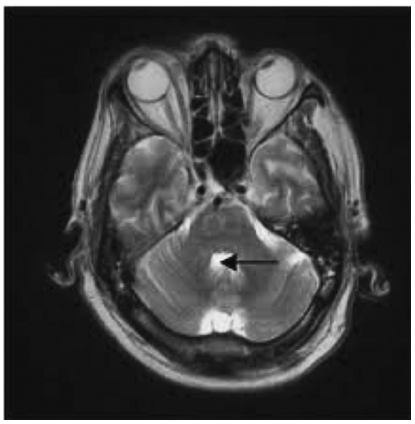

Fig. 3. T2 - weighted image of hyperintensity in the pons (black arrow) consistent with CPM (day 14).

A 58-year old man presented to M.B.S Hospital, Government Medical College, Kota, Rajasthan, with non-projectile vomiting and watery diarrhoea for the last three days. On admission, his initial blood tests were within normal range except for severe hyponatremia with a sodium level of 100 mmol/l (normal, 135-145 mmol/l). Normal saline was administered intravenously, and the sodium level began to normalize at 100, 106, 122 and 130 mmol/l on days 2, 3, 5 and 7, respectively. One week later, he had complaints of difficulty in swallowing and speaking accompanied by unsteadiness of gait and eventual inability to walk. Initial magnetic resonance imaging (MRI) (Fig. 1) and computed tomography (CT) of the brain on day 7 showed no abnormality. However, his condition continued to deteriorate, and a repeat MRI on day 14 revealed a well-defined lesion in the pons of low T1 signal intensity (Fig. 2) as well as a lesion of high T2 signal intensity (Fig. 3). A diagnosis of central pontine myelinolysis (CPM) was made.

CPM is an subacute demyelinating condition of the brain stem and a recognized complication of the treatment of patients with chronic hyponatremia (>48 h)[1]. The risk of CPM is believed to be associated with a rapid (>8 mmol/l/day) correction or overcorrection of the serum sodium concentration. However, there is no accepted safe rate of correction[2]. Conventional imaging findings (MRI and CT) lag behind clinical manifestations, thus limiting the utility of imaging in early diagnosis. Imaging is advocated later to confirm CPM diagnosis[3]. There are no effective therapeutic methods for CPM treatment and recovery varies ranging from none to substantial improvement[4]. Although the use of plasmapheresis (PP) and immunoglobulins (IVIg) has been reported in analogous situations, support for their use in the liver transplant population is limited but, there is no clear evidence as to why PP and IVIG may help reverse the neurologic sequelae of CPM. However, it has been suggested that the initial osmotic

stress results in the release of myelinotoxic products that can lead to demyelination[5-7].

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